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VASCULAR TENSION IN CHRONIC ILLNESS.

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THIRTY years ago, the study of disease of the vascular system scarcely did more than cover the lesions of the valves of the heart, a consideration of gross lesions in its muscle, and a brief reference to the grave changes found in the bloodvessels when they were affected by marked fibrosis or atheromatous change, or when they were plugged by emboli or thrombi. Little attention was paid to the changes in the finer arterioles except when these changes were well developed, and nothing was said of the functional state of the arterial system in its relation to circulatory disorders. Nearly all the treatment advised was directed to the heart itself, and the importance of the vasomotor system as a factor in disease was almost ignored. At the present time, however, medical men in general have come to recognize the fact that a vascular system intact, both functionally and anatomically, is essential not only to the health of the body in general but of the heart in particular. Not only may serious consequences to the heart follow the development of arteriocapillary fibrosis, but, in addition, mere spasmodic contractions of the arterioles, to a degree in excess of the normal, may be disastrous in acute conditions; and the antithesis of spasmodic contraction, namely, vascular relaxation, may result in speedy death, either by bleeding the man into his own vessels, or by causing cardiac exhaustion in the endeavor of this organ to fill the relaxed vessels. The object

of these remarks, therefore, is to emphasize the importance of studying the vascular state and the degree of the blood pressure in respect to the diagnosis, prognosis, and treatment in several states of disease, and they are called forth because a fairly large experience has convinced me that many medical men are still impressed with the idea that a tired heart should be stimulated to greater effort when in reality it should be given as much rest as possible by diminishing its burden. I believe that this is one of the grave mistakes in modern diagnosis and treatment.

If we take, as an example, any one of the grave infectious diseases characterized by toxæmia, we find that the heart fails, if it fails at all, as a result of poisoning of its muscle or disorder of its nerve supply arising from the same cause, and against this muscle poison our stimulants do not act as true antagonists. The longer I study these cases the more I am impressed with the fact that active cardiac stimulation is often more harmful than advantageous, unless we can resort to measures designed to diminish the toxæmia, and by decreasing the toxæmia permit the heart to do its work. As a matter of fact, the vascular system in these cases shows, if it be carefully studied, that it is much more responsible for the circulatory depression which is present than is the heart, and an examination of the patient's vessels will reveal the fact that they are widely dilated, and of the heart that it is working more actively and more forcibly than in health in an endeavor to fill the relaxed arterioles. The patient's death is doubtless imminent because of circulatory failure, but the heart, in a considerable portion of cases fails only when it is tired out in the vain attempt just alluded to. To use a simile which I have used before, the heart under these circumstances may be compared to a locomotive on a slippery track. It fails to push its load not because it is weak, but because it finds no resistance on the rails. The more the locomotive is stimulated to increased endeavor by opening the throttle wider and wider, the more it shakes itself to pieces, the more it expends its energy, and it gains nothing. The heart is designed to beat against a very considerable arterial tension, just as the locomotive is required to work against a given load; but here the simile ceases, for the locomotive gathers no strength from the friction or resistance of the rails and train, whereas the heart depends absolutely for its existence upon such resistance, because if the arterial pressure falls the heart muscle gets less blood through its coronary arteries and through the vessels of Thebesius. A very low tension, therefore, means an increased but worthless expenditure of energy and a decreased nutritive supply to the overworked muscle fibres. Fortunately most of the drugs commonly used by physicians under these conditions are, as a matter of fact, both cardiac and vascular stimulants, and so the patient gets the needed vascular effect.

It is quite remarkable how competent the heart is to attend

to its functions if the physician is able to overcome vascular relaxation. There are only three drugs which have seemed to me to be competent for this purpose, namely, atropine, adrenalin, and digitalis, and I have placed them in their order of usefulness: for atropine by contracting the great vascular areas of the abdomen raises arterial pressure everywhere and, according to Hedborn, dilates the coronary vessels, and so increases the cardiac blood supply without directly stimulating the heart muscle to increased endeavor.

Two states of deleterious high tension are also overlooked, namely, the high tension of acute illness and the high tension of prolonged nervous and mental stress. The high tension of cases of acute illness rarely approaches the high tension characteristic of arteriocapillary fibrosis for obvious reasons, and so is not so readily recognized by the physician who is careless of this aspect of his patient's case; but, nevertheless, it is important for us to recall that its modification is much more advantageous than the very popular efforts to reduce fever, which is an associated symptom of comparatively little importance. Whether the high tension occurs early in the illness or at a later stage, vascular relaxants, which are not cardiac depressants, will save the heart from the expenditure of a large amount of unnecessary effort, and thereby enable it to withstand calls upon its reserve energy.

I wish to call attention to only two more points in regard to this matter of high arterial tension in acute disease, namely, that physicians often, in their terror of cardiac failure, give circulatory stimulants when a comparison of the patient's pulse force and tension with that in their own vessels will show that their own circulation, although normal, is at a lower ebb than the patient's, and by giving stimulants when fever is already causing excessive cardiac and arterial activity, they place themselves in the position of the man who beat his horse into a run for fear he would give out before he got to the end of the journey.

The second point is that our forefathers, while they were empirics, were often more skilful in these cases than many so-called scientific moderns, for they gave vascular relaxants in the form of spirit of nitrous ether, which is, after all, like nitroglycerin in its effects, and Dover's powder, which produces similar influences.

The subject of arterial spasm or fibrosis in its relation to cardiac failure, in those who are subjected to mental strain, is too large a field to be discussed at this time, although it is more important than any of the fields already spoken of. I have recently discussed it at length elsewhere.¹

To express the matter briefly we meet with three classes of cases of high arterial tension of this type: (1) Those in which persistently high tension is a result of spasm due to prolonged nervous stress

¹ Therapeutic Gazette, December, 1905.

combined with certain abuses as to habits of life, food, and drink. (2) Those in which tension is high because in addition to spasm there is gradually developing, or has already developed, fibroid changes in the vessels. (3) Cases in which, after a prolonged period of high tension, there, more or less suddenly, develops persistent low tension in which the arteries are relaxed and distended, so that, to some degree, they resemble veins in their calibre and compressibility.

In the first and second classes rest in bed, with massage and the nitrites, is essential. In the third class the rest in bed is needed also, but vascular stimulants are required. In all these cases the iodides are valuable. If the high tension is due chiefly to spasm, nitroglycerin will do good for obvious reasons, but it will fail to a great extent if the condition is due to fibrosis. This is an important therapeutic point, showing that the physician can expect nitroglycerin to reduce only that part of the hypertension which is due to spasm and not that part due to fibroid change. The latter stage is largely beyond the influence of medicinal agents, unless it be the iodides. On the other hand, the combined effect of rest, massage, and iodides will often, even when fibrosis is well developed, not only produce excellent results, in that the state of a tired heart is improved, but also result in a marked diminution of arterial tension, partly by a cardiac and partly by a vascular influence.

There is still another class of cases in which high arterial tension is present which deserves notice, namely, those persons of somewhat advanced years who have gradually developed a high tension of moderate degree, and to this tension the heart, vessels, and tissues have become adjusted, so that it has become a "standard tension," so to speak. With the onset of an acute illness the physician may find this standard tension somewhat exaggerated, and, impressed with the general principle that a high tension is evil, he proceeds to lower it to normal instead of to the new normal standard of the individual under observation. This is usually unwise, and each patient must be studied carefully in this respect.

Finally, let me sum up the points which I am desirous of presenting to the clinician:

1. Cardiovascular stimulants are often given when vascular relaxants are really needed.
2. More attention to the protection of the heart from unnecessary labor is advisable.
3. If the physician will direct his treatment to the vessels the heart will often be able to care for itself.
4. Do not stimulate a heart to increased effort when the real object is to decrease its burden and to diminish the toxæmia which is destroying its function.